Modern Concepts of Cardiovascular Disease

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CHRONIC CONSTRICTIVE PERICARDITIS: RESULTS OF PARTIAL PERICARDIECTOMY AND EPICARDIOLYSIS IN THIRTY-FOUR CASES

The surgical treatment of cardiac lesions has developed more slowly than other fields of surgery because of the difficult problems which these lesions present and the hazard associated with their operative treatment. The problems in this field of surgery are concerned not only with technical surgical consideration but also with the diagnostic difficulties encountered, and it is only because of the close co-operation of the clinician and the surgeon that progress in this field has been made possible.

One of the important advances in the surgical treatment of cardiac lesions in recent years has been made in the treatment of cardiac tamponade associated with chronic constrictive pericarditis. Galen and Morgagni recognized the condition at necropsy but Lower is given the credit for the first clinical description of the disease in 1669. In 1895, Weill suggested that the condition would come within the province of surgery by delivering the heart from the shell which strangles it. He stated that after the adhesions reach a fibrous stage they act independently of their original cause and that medical therapy is illusory. Délorme, in 1898, proposed an operation for separation of the adhesions between the heart and the pericardium either by sharp or blunt dissection. Although he made many pleas for this type of operation before the clinical societies of Paris, this procedure was not carried out until 1913 when Rehn followed Délorme's proposal and operated on four patients with constrictive pericarditis. Two of the patients died at the operation and the remaining two died within the next two years. Rehn not only followed Délorme's suggestion of separating the adhesions but also resected a portion of the pericardium. Sauerbruch, who worked independently in the same year, reported a case in which partial pericardiectomy was successful. Brauer in 1902 suggested removal of the precordial bony structures so that the tug of the heart would be on the soft yielding structures of the thoracic wall. This procedure is only effectual in cases in which the adhesions involve the pericardium and thoracic wall and the pericardium is not adherent to the heart muscle; it offers little if any benefit for true constricting pericardium which compresses the heart muscle. When this condition exists, the actual removal of the constricting scar from the heart muscle is the only effectual procedure.

The work of these pioneers in establishing the fact that constrictive pericarditis was amenable to surgical treatment has stimulated clinical studies and investigative work in this field.

Schmieden, of Germany, and Beck, Churchill and Blalock in the United States, have done a great deal of work on this condition and have had considerable experience with its surgical treatment.

Many different terms have been used to designate cardiac tamponade, such as "adhesive pericarditis," "Pick's syndrome" and "chronic constrictive pericarditis"; however, the last term is the one most generally accepted to designate an inflammatory lesion of the pericardium and epicardium in which fibrous adhesions often associated with deposits of calcium and occasionally pockets of encapsulated fluid form between these coverings of the heart and in which the inflammatory scar contracts around and onto the heart muscle to such a degree that it interferes with the normal diastolic and systolic functions of the heart and causes impairment of circulation. This condition is of unusual interest because its cause is often obscure, its occurrence is infrequent, its clinical course is slow and it causes progressive disability.

The circulatory failure produced by this condition develops slowly and insidiously. It is due primarily to progressive mechanical interference with the action of the heart and not to intrinsic cardiac disease except in those cases in which it has been present for a long time and some degree of associated myocardial atrophy has resulted from the constriction. The primary cardiac fault in this condition is physiologic, owing to the fact that the dense inflammatory scar encasing the heart muscle prevents the heart from attaining its normal diastolic volume and thereby causes increased tension in the chamber of the right side of the heart which results in an inflow stasis.

The diastolic filling of the heart is less than normal, which results in decreased ventricular output of blood. It is probable that other factors contribute to the decreased ventricular output to a lesser but significant degree. One of these factors is interference with systolic contraction of the ventricles due not only to fixation of the scar to the myocardium but also to myocardial atrophy and degeneration from limited action as well as from the original infectious process.

The primary infectious process which causes constrictive pericarditis may not be and often is not associated with acute symptoms which indicate that the pericardium has been involved.

The cause is of considerable interest and importance and there is some difference of opinion concerning the type of infecting organism. Every effort was made to determine the causative agent in a series of thirty-four cases in which I operated. This study definitely proved that the etiologic agent was tuberculosis in five, or 15 per cent, of the cases. This type of infection, therefore, should always be considered as a possible cause of the disease. In twenty-nine, or 85 per cent, of the cases, the type of primary infection was unknown.

It has been stated by some authors that rheumatic fever is one of the chief causes of this disease but it is of interest to note that in only one of the cases was there a previous history of rheumatic fever and in this case the microscopic study of the tissue removed at operation revealed that tuberculosis was the etiologic agent. I do not believe it possible or advisable to attempt to draw any definite conclusions as to the cause of the disease from this relatively small group of cases.

The two most common conditions with which constrictive pericarditis is most likely to be confused

clinically are cirrhosis of the liver and congestive heart failure due to intrinsic cardiac disease. In most instances, however, the sequence and insidious onset of symptoms, when they are correlated with the physical and laboratory findings, are sufficiently characteristic to make a definite diagnosis of constrictive pericarditis.

The most important clinical and laboratory findings in the thirty-four cases were increased venous pressure, prolonged circulation time, enlargement of the liver with impaired function, ascites, faint heart sounds and diminished pulsations, infrequency of cardiac murmurs and low blood pressure and pulse pressure. The heart usually was of normal size or slightly enlarged but was not dilated.

The number and severity of subjective symptoms increase progressively with the course of the disease. The effects of the disease cause increasing disability which gradually becomes complete unless relieved by surgical treatment.

Surgical Treatment

Patients who have been selected for surgical treatment require pre-operative preparation. Measures to reduce the ascites and the peripheral edema and to improve the function of the liver comprise the chief objectives in pre-operative treatment.

I prefer general anesthesia produced with cyclopropane in combination with some ether administered under positive pressure by the use of an intratracheal tube. In my earlier cases I used cyclopropane only. The reason for combining it with ether is to lessen the occurrence of cardiac arrhythmia, which may occasionally be associated with cyclopropane anesthesia.

During the operative procedure it is important not to open the pleural cavity, particularly at an early stage of the operation, because of the respiratory difficulty that often follows. I defer the wide separation of the left pleural attachments to the outer wall of the pericardium until after the pericardial scar has been fully separated from the myocardium.

The most important part of the operative procedure is the release of the myocardium from the constricting scar. I have suggested the term "epicardiolysis" to designate the separation of the scar of the innermost layer of the pericardium from the myocardium. This procedure is very important; it is done in addition to the resection of a portion of the pericardium and epicardium and extends over a much larger area of the heart muscle than the area from which the pericardial scar is resected.

Opinions differ concerning the amount of pericardial scar that it is necessary to remove as well as concerning the amount of scar to be separated from the heart muscle. I believe it is advisable to separate the scar from the ventricles and as much as possible from the right auricle and orifice of the inferior vena cava. It is of particular importance to sever the attachments of the right ventricle to the diaphragm. It is of equal importance to separate the apex of the heart from its attachments and I believe that this should be done early in the operation, if possible. Separation of the heart muscle from its fixed attachments to the diaphragm is one of the most important considerations in re-establishing the action of the heart. Improvement in the function of the heart often is noted immediately after adhesions of the apex and right ventricle to the diaphragm have been severed.

After the scar has been separated from the heart muscle, the amount of pericardium to be resected depends on the character of the scar and to some extent on the fixation of the pleural attachments to the outer wall of the pericardium. I believe it is advisable to resect as much of the anterolateral wall of the pericardium as possible without injuring the pleura. By careful dissection, the pleural attachment to the pericardium can be separated to approximately the position of the left phrenic nerve, and the entire anterior portion of the thickened pericardium extending beneath the sternum is resected. In cases in which attachment of the pleura extends high on the pericardium, as it often does when calcium is present, it may be impossible to resect an adequate amount of pericardium without injuring the pleura. In these cases, linear incisions into the remaining cut edges of the pericardial scar will produce a much wider opening of the pericardium.

The importance of separating and removing sufficient scar to release the heart cannot be overestimated because, if a sufficient amount is not separated and removed, cardiac action will continue to be impaired. This not only will eliminate the possibility of ultimate recovery from the disease but will interfere with the immediate recovery from the operation.

Results

The rapidity and completeness with which recovery occurs after operation depend on several factors, the most important of which are the amount of myocardial atrophy and degeneration, the amount of hepatic damage and the thoroughness with which the constricting scar has been removed from the impaired heart. Improvement may be noticed soon after the operation but it often proceeds slowly and progressively. Complete recovery may not occur for many months. This is somewhat dependent on the duration of the disease before operation.

Of the thirty-four patients operated on, twenty-seven recovered from the operation and seven died in the hospital after operation; the operative mortality, therefore, was 21 per cent. Although this mortality is relatively high, it is commensurate with the seriousness of the disease and with the fact that the mortality rate ultimately would have been 100 per cent without surgical intervention.

Eighteen of these thirty-four patients can be considered cured at this time and two patients, who have been operated recently, six and three months ago respectively, have shown marked progressive improvement. Of the remaining seven patients who recovered from operation, three have shown a moderate degree of improvement but it is not likely that they will ever be considered cured. Four patients have died since the operation. The deaths of three of these four patients were due to continuation of the disease; two of the three deaths were due to progressive cardiac failure two years and one month, and seven years and six months, respectively, after operation, and one was due to tuberculous peritonitis two years and three months after operation. In the remaining case, the patient was improving satisfactorily from operation but unfortunately died of pneumonia seven months after the operation.

I believe that the percentage of deaths can be decreased by earlier recognition of the disease, which would permit earlier institution of surgical treatment.

Patients who respond to operative treatment for chronic constrictive pericarditis obtain one of the most dramatic results produced by operation and it is most gratifying to see these patients, who otherwise would be doomed to a slow lingering death, restored to health and usefulness.

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